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IDIOPATHIC STATUS HEMI-EPILEPTICUS AND GENUINE FOCAL EPILEPSY[1]

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In bringing the subject of localized convulsions or rather epiteptoid convulsions before you, I wish to confine the scope of the paper to that group of cases which are of genuine epileptic character in contradistinction to symptomatic epilepsy of the Jacksonian or reflex type.

The material for this paper is partly anatomical, but chiefly clinical, and I feel that there may be two opinions as to the underlying pathological changes in the cortex of the brain in these cases. I have, however, been very careful to choose, after a most minute examination, only such cases in which the physical examination has been entirely negative.

If there was a history of trauma with unconsciousness, even through there was no fracture of the skull, I have ruled out the case.

If the previous pathological history was such as to point to the possibility of an encephalitis during the course of an acute infectious disease, the case was not included in the group. We have been careful to enquire into the presence of congenital or acquired syphilis. All cases which presented any deviation from the normal, such as slight degree of ptosis, inequality of the pupils, difference in the muscular innervation of the two sides of the face, tongue and body, changes in the reflexes, no matter how slight, even though no relation could be shown between the attacks and the supposed lesion producing the above slight deviation, were not included, because of the suspicion of the presence of an organic lesion in brain or nerve trunks.

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[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

I have known these cases from a few years up to fifteen years, wherever possible, have watched them up to the present time, and I feel that I can safely say that as far as our methods of physical examination go, I have ruled out all cases with apparent cortical lesions.

I would like to categorize these cases as cases of genuine epilepsy in the sense that the attacks are of unknown origin in contradistinction to those cases which are symptomatic. These cases are of interest in a two-fold manner; first, as a study of one of the manifold phases of epilepsy, and secondly, from the standpoint of the question of surgical intervention.

The cases comprised under this group can be arranged into two classes:

1. Hemi-epilepsy and more especially the status hemi-epilepticus.
2. Localized epileptoid seizures in the form of partial convulsions or the initial manifestations on the part of the special senses or of the power of speech.

Binswanger defines genuine epilepsy as a "well-defined chronic disease of the central nervous system, which can be produced by manifold causes. It manifests itself either by frequent attacks of convulsions with unconsciousness or in partial attacks of this kind or in psychopathic states occurring either in or after the attacks."

There is some question whether some of the cases of status hemi-epilepticus can be conformed to the above definition of epilepsy.

Müller was the first to call attention to a status hemi-epilepticus, which ends fatally without any discoverable lesion of the brain or its cortex, and named the condition status hemi-epilepticus ideopathicus. The condition is not very frequent and there are but few cases on record. Of these there are two groups, either the patient has never had any convulsive attacks before or the hemi-epileptic status occurs intercurrent in a case of genuine epilepsy, or the case, if it recovers, subsequently becomes one of general epilepsy.

Most cases on record have terminated fatally, usually after surgical interference, and the autopsy revealed no cause for the hemi-epileptic character of the convulsions.

The following case may serve as a type for this form of localized convulsions:

CASE No. I.—Status hemi-epilepticus. M. F., æt. 22. Female, servant—parents are nervous. Father alcoholic. Mother neurasthenic. Brother and sister are well. No epileptics or insane in any branch of family.

Was injured at age of seven, struck on head, no loss of consciousness, right leg seemed weak for some time afterwards. Has never been sick afterward and never had a convulsion.

Grew up to be a very strong, muscular woman.

Has been well absolutely and a hard worker, never sick, no headaches. Eight days before death arose in the morning with a violent headache; this soon passed off, and patient felt well all day until she retired at 9 P. M., when she began to vomit and had a chill; vomiting continued at intervals for two hours. Patient then fell asleep and was found unconscious next morning.

A physician was called and gave her a hypodermic of strychnine; after regaining consciousness in half an hour she was unable to speak, felt drowsy, fell asleep easily. Pulse was weak, temperature was normal. At noon she felt well. All next day and the day following she was absolutely well, attended to her household duties, cleaned windows, cooked dinner, etc.

On fourth day arose feeling well up to noon, then had vertigo, sinking spells, fear of losing mind and seemed somewhat incoherent in her speech.

On same night she had a convulsion with loss of consciousness. On fifth and sixth days no convulsion but patient seemed half dazed, staggered about the house, complaining of weakness and vertigo. Forty-eight hours preceding death patient was in convulsions continually and did not regain consciousness.

All convulsions began on the right side of face, then extended to right arm and right leg, after which they became general. The convulsions lasted about two minutes and were followed by a complete paralysis of the right side of body, face, arm and leg. In the interval between convulsions patient was restless, tossed about with left arm and leg, gradually movement began, after the lapse of a few minutes, in the right arm and leg and these movements became more and more marked until the muscular activity of the right side seemed entirely restored before the onset of the next convulsion. The patient was observed by me twenty-four hours before her death and in one half hour I observed three convulsions.

The physical examination showed a well developed, strong young woman, of good height and in good flesh, no edema anywhere.

Mental Condition.—Entirely unconscious and could not be aroused. Pupils equal in size, reaction to light sluggish between attacks. Papilla somewhat injected. Passing paresis of both arm and leg of right side, reflexes abolished on both sides.

Slight amount of albumin in urine with some granular casts. Temperature normal, pulse 90. Patient was removed to a hospital, but after a surgical consultation, no definite reason could be assigned for surgical intervention except the Jacksonian character of the convulsions. The acute character of the case and the status epilepticus seem to be contraindications to an operation.

Patient died on the following day, the convulsions diminishing in frequency toward the end.

Autopsy.—Brain. There was an intense venous congestion over the entire surface of brain, more marked on left side than on right. Especial attention was paid to the various sinuses in the dura-mater but all were found to be normal. The middle cerebral artery on both sides and all their branches, especial care being paid to the left artery, as far as the ramifications could be followed were found patulous and normal.

The cortex of the brain, after a most minute examination, especially over the left psychomotor areas, was found to show no macroscopic changes; punctate hemorrhages and small areas of softening were looked for, but none were found. The pia mater showed no adhesions anywhere. The dura mater was normal. The sections of the cortex were misplaced and no microscopic examination was made.

The kidneys were macroscopically normal, microscopically slight fatty changes were found in the cells.

In a word, the autopsy was negative. The kidney changes were probably secondary to the general condition, not the primary cause.

Here we have a case where the entire previous history was known.

The first attack, one week previous to her death, came on during sleep, the patient being found in a comatose condition from which she recovered in a few hours, then went about her work for two days. The attacks then coming on again, first few in number, with conscious intervals of hours during which the paresis of the affected limbs disappeared, then a gradual increase in the number of attacks until there was no return of consciousness between the attacks; finally the attacks became so numerous that she practically passed from one attack into another.

There can be no doubt but what some general intoxication produced all the symptoms, including the albuminuria; that this condition was entirely uremic is doubtful. We see similar general intoxication, say, for instance, of la grippe, followed by a general infection, including the albuminuria with granular casts, and we have no uremic manifestations at all, to say nothing of the convulsions.

Numerous similar cases are on record without the albuminuria. Then, again, the localized convulsions in uremia are not of the same character and the cases do not yield to the treatment for uremia. In this particular case the kidneys did not show the changes of acute parenchymatous nephritis. On the other hand, the important points under consideration are the half-sided convulsions and the negative autopsy, leaving the cause for the half-sided convulsions unexplained. The injury at the age of seven, with a doubtful paralysis of the right leg might, before death, point to some old scar in the left leg area, but the convulsions always began in the right side of face. Again, the cortex in the left leg area was found normal macroscopically. The entire illness lasted for nine days, and if any gross lesion had caused the half-sided convulsion, even though they had been punctate hemorrhages or minute areas of softening in the sense of Spiller and McCarthy, they should have been visible to the naked eye.

Cases of this kind belong in a category by themselves. It is true that in their mode of origin, the spread of the convulsive seizure and the secondary paralysis following the return of consciousness or in the restless period between the convulsive seizure when patient was comatose, and the rapid disappearance of the paralysis were in a very close resemblance to the ordinary Jacksonian epilepsy. But here the resemblance ceases. In ordinary Jacksonian epilepsy, except in the acute traumatic cases, I have never seen a condition of status epilepticus; moreover, the previous histories of the cases point to a gradual onset over a period of months and even years, whereas all the recorded cases of status hemi-epilepticus usually have a history of acute sudden origin and a rapid fatal termination or an equally rapid recovery in the few instances where recovery has occurred. There are quite a number of these cases on record; I mention those of Bernard, Bonnhoff, J. S. McKendrick, and especially Leononere and Henneberg.

McKendrick's case is interesting on account of the fact that his patient had numerous attacks without loss of consciousness, limited usually to right side, followed by aphasia, paresis and hemianesthesia, in one attack the left side alone was involved. This patient died in status hemi-epilepticus and the autopsy was absolutely negative, both macroscopically and microscopically.

Leo Müller reports a series of eight cases of this character with autopsy negative both for macroscopic and microscopic examination.

In each of these eight cases no evidence could be found that the individuals were formerly epileptic; all died in a state of status hemi-epilepticus with negative autopsy. Müller, after a review of the literature, comes to the conclusion that in both of these so-called cases of Jacksonian epilepsy as well as in ordinary genuine epilepsy we can have a status epilepticus which terminates fatally. This does not, however, explain the case; it merely states a fact.

That we might have minute foci of inflammation and degeration throughout the cortex, as is shown by Spiller, McCarthy and others for uremic cases, would hardly explain the cause of death, nor would the finding of such foci explain the hemi-epileptic character of the seizures; they themselves are merely the effect and not the primary cause; we might suppose an embolic invasion of the various branches of the arteria fossa sylvii so numerous and minute as to effect a widespread area on one side of the brain. But in our case, at least, where a week elapsed between the first attack and the fatal outcome, we would suppose that macroscopic evidences of minute hemorrhagic or degenerative areas ought to be found.

It is possible, too, that these cases represent suddenly developed acute epilepsy which rapidly passes to a state of status epilepticus and death.

The half-sided attacks could be caused by the fact that one hemisphere is more vulnerable, functionally weaker than the other and that as a result of a toxemic condition the irritations begin in the weaker side of the brain in the form of localized convulsions which rapidly spread and end in a general convulsion. We have an analogy for this explanation in those cases of genuine epilepsy in which the general convulsion is followed by monoplegia, hemiplegia, aphasia, deafness, etc., which quickly disappear after the attack. These secondary manifestations are explained by an excess of exhaustion in the corresponding cortical areas. There must, however, be some local weakness of these centers, which explains this excess of exhaustion. In a similar way we might explain that the half-sided origin and nature of these convulsions may be due to a functional weakness of one hemisphere compared to the other.

The most important lesson, however, that is taught us by the study of above and similar cases of status hemi-epilepticus is the futility of surgical interference. In fact, the very character of these seizures, that they have all the ear-marks of Jacksonian epilepsy, that they occur in individuals whose previous history has been negative, and the rapid recurrence and number of seizures must be looked upon as rather indications against surgical interference than for such procedure.

There is an air of mystery about these cases which only future investigation can clear up.

I have seen three other cases of half-sided convulsions, which have had at times so many convulsions in twenty-four hours that we can speak of a passing status epilepticus. In one of these cases there was never any loss of consciousness, but in the other two loss of consciousness occurs during the night attacks, especially if the attacks during the day have been very numerous. The question of hysteria can be raised in all three cases, but in two of the cases the loss of consciousness with secondary sleep and amnesia and especially the fact that the patients are aroused from deep sleep by the localized convulsions and become unconscious after the involvement of the whole side, would speak in a positive manner for epilepsy rather than hysteria. One of the last-named patients is the sister of a physician, who vouches for the fact that she is really unconscious, and the other has been observed repeatedly by nurses in a hospital under my care.

The first case whose history I give in a concise manner is the son of a physician and may seem a little doubtful. The history is given by the father.

Alfred B., æt. 21. Had never had any of the ordinary diseases of childhood. Parents are well. At four years of age, began to have spasms in the left arm, which continued daily, and after several months increased in extension until the entire left side, including the face, became involved, at times he has had as many as twenty attacks per day. Patient never has less than eight to ten attacks per day; has frequent attacks during the night. At the end of one year the left side of body was paralyzed. Patient, six years of age, was subjected to anal dilatation (orificial surgery!!!), was isolated in a hospital and after two months the attacks gradually subsided and the paralysis disappeared. Never any loss of consciousness.

During the next seven years patient was free from attacks.

During the past eight years patient has had frequent localized convulsions in the left arm, without any paralysis and without loss of consciousness.

Examination.—Patient is a tall, handsome, well-built athletic young man. Mental condition good, is bright and cheerful, never has any headache or vertigo. Stands well in his class at Yale and is fond of all out-door sports. General muscular development good. No change in optic disks. Pupils equal in size and react to light. There is an absence of both patellar reflexes, otherwise the examination for muscular power, sensation and reflexes is negative. After the examination patient was seized with a spasm of the left arm, tonic in character, somewhat painful, the fingers were flexed, the hand almost closed and flexed on the forearm, the forearm in supination and bent upon the arm, the biceps also involved in the spasm. Duration about one minute. There was no weakness after the spasm subsided.

Patient was given twenty grains of strontium bromide at bedtime and took the medicine for one month and has had no return of the spasm for the past twenty months.

Résumé.—Onset at five years of age, spasm of left arm, gradually involving the whole left side, with paralysis. Patient has as many as twenty attacks per day; after fourteen months of treatment was completely cured by dilatation of anus and a rest cure without use of any drugs. Recurrence of local spasm in the left arm after seven years, countless attacks during the next eight years and then sudden cessation after the use of twenty grains of strontium bromide at night for one month.

I have seen the other two cases of a similar character, without, however, the happy result, grow up from childhood to womanhood with constant attacks of a hemi-epileptic character without permanent paralysis, with loss of consciousness occurring occasionally and yet without the usual stigmata of hysteria.

CASE III.—Miss M., æt. 7, of Hebrew parentage. The child has always been well, has not had any serious illness with the exception of whooping cough. About two years after the whooping cough she began to have fainting spells for which there was no adequate cause.

Present trouble began at the age of five with spasms in the right hand which extend to the leg and are painful in character.

During the attack patient becomes very nervous, is unable to speak, but does not lose consciousness except on rare occasions. The attacks occur often during the day; frequently during the night the patient is awakened from her sleep by the attacks; the attacks last but a few minutes and are not followed by weakness in the arm or leg. On rare occasions patient will not have attacks for a day or two at a time, but the rule is to have numerous attacks daily.

Examination.—Mental condition only fair. General development good. No loss of muscular strength, no anesthesia on right side, reflexes normal, special senses normal.

During the past thirteen years she has grown to be a large, well-built woman, suffers no headaches or vertigo. Is never ill, but the local spasms of the right side of body continue. The right side of body has grown and developed as well as the left, and there has never been any paralysis. Patient at times loses consciousness, especially so during night attacks, when the number of attacks during the day preceding have been especially numerous. In one attack the patient fell and sustained a Colles fracture. The attacks in the arm continued during the time the fracture was in plaster of paris.

Her mental state is somewhat backward, due in a measure to the fact that she has never been able to attend school.

A similar case in every respect is the following:

Bessie C., æt. 26, small town, Indiana. Family history negative. Fourteen years ago had a fall. Since then has not been well. Did not strike head and did not lose consciousness. She says that her right arm and leg were weak periodically, before the onset of the convulsions, for years after the fall, before the convulsions began. Patient has attacks of convulsions which begin in the right arm, then extend to the right leg and right side of face, duration only a few minutes. Usually attacks pass off in a few minutes without loss of consciousness, but has lost consciousness on several occasions, but very rarely. The attacks are followed always by a weakness of the right arm, but not always of the leg and never of the face, which quickly passes away. These attacks occur with great frequency during the day, attacks with loss of consciousness occur only at night. Patient is awakened from her sleep with a spasm in the right forearm or the right foot. The spasm is at first tonic, then clonic, very severe and painful; in a few seconds the entire right side is affected, patient is unable to talk or cry out and then loses consciousness, from which she awakens in an hour or two, has a bad headache, feels badly all next day.

In the attacks observed at the hospital there has been no convulsion at all on the left side during the period of unconsciousness. After the attack patient at times arises, walks about, does automatic acts of which she afterward has no recollection.

Most of the spasms are limited to arm, leg and face; they are tonic in character for a few seconds, then clonic, the spasmodic contractions continuing for several minutes, often so slight that they can hardly be seen, but can be distinctly felt if the arm is grasped. The initial tonic spasm is always severe.

There is no marked headache, no vomiting and no vertigo.

For days at a time patient passes from one paroxysm into another. These are of the character described below. Each attack, both clonic and tonic, does not exceed two minutes in duration. At times the attacks are as numerous as ten per hour, usually beginning in the arm, but at times simultaneously either in arm and foot or face and arm. Then for days at a time the right side is entirely free from attacks.

Bromides reduce the number of seizures, but the above condition has continued now for ten years. Patient can attend to the ordinary household duties on a farm.

Examination.—A well-developed and well-nourished woman of medium height. Has a bromine eruption all over face and back.

Mental condition good, memory quick and active.

There is an exaggeration of the reflexes but no evidence whatever of any organic disease of the brain.

Résumé.—This case has gone on in about the same way for the past fourteen years, spasms numerous, in character always beginning in the right arm and extending to leg, with at times a passing paresis and at times a loss of consciousness, but neither the history of the case nor careful observation reveals the presence of any organic lesion of the cortex. The fact that the severe attacks with loss of consciousness occur at night and that patient is aroused from her sleep by the spasms in the arm or foot proves that this case is epileptic rather than hysterical in character. It is rather a good point in differential diagnosis between genuine epilepsy on the one hand and Jacksonian on the other, that the local spasms in the arm awakens patient to consciousness out of a deep sleep before the convulsion has had time to become general on the right side and before consciousness is lost. But all proof of an organic lesion for the Jacksonian seizure is wanting.

I have no hesitation in designating these three cases as epileptic and also believe that the numerous attacks, in one case continuous for days at a time, constitute a condition of status hemi-epilepticus, even though there is no continuous loss of consciousness. The question as to the functional character or organic origin of these attacks in these cases will be taken up later.

The question as to whether a localized convulsion, limited to one group of muscles, followed by paresis and aphasia, is really due to a cortical lesion is often very difficult to decide at the time of the first examination. We must often wait for weeks and even months, even with such gross lesions as tumor or syphilitic meningitis, before a second attack occurs.

Our knowledge on the subject of Jacksonian epilepsy is undergoing some rearrangement. The line of division between convulsive seizures of a functional character and those due to an organic lesion of the cortex is not as sharp as it might be.

The opinion is gaining more and more ground that there is a goodly number of borderland cases in which it is difficult to say even after a long study of the case, whether the localized convulsion is caused by an organic lesion, whether it is to be looked upon as a motor aura, whether it itself is to be classified as genuine epilepsy, or whether it is purely a reflex contraction, or whether it is hysterical in character. We must also remember that every Jacksonian attack need not be necessarily caused by a gross lesion of the cortex of the psychomotor area. Diseased conditions of the vessel walls, local circumscribed changes of the bony or membranous covers, subcortical scars, scars or areas of softening in the neighboring parietal or the under surface of the frontal lobe can cause Jacksonian seizures, if other causes such as alcoholism or the various intoxications have been at work to produce an epileptogenic condition of the cortex. Even atrophic conditions of the cerebellum have been found as the only cause of Jacksonian convulsive seizures. We can see therefore how difficult it is in a given case of localized epileptic seizures, to rule out organic causes as the underlying condition of the attack. When we consider that there are brain lesions which may occur during the embryonic period, slight attacks of cortical encephalitis producing temporary delirium during the acute infectious diseases of early childhood and which may disappear without leaving a scar beyond the slight changes in the ganglionic cells of the cortex, but which may nevertheless be the point of origin of cortical irritation leading to Jacksonian attacks, we can readily understand the position which has lately been taken by numerous authors, Freud and Rie, Rosenberg, Nurerricht, Koenig, Murutoff, Lukacz, Starr, Sachs, Pick, Heilbronner, etc., who hold that epilepsy is an organic disease of the cortex. The advocates of the organic theory of the origin of epilepsy have not been without their opponents, prominent among whom have been Oppenheim and Binswanger. While they admit that there may be an organic basis for epilepsy, they maintain that the latter has not as yet been demonstrated and that the distinction between genuine and symptomatic epilepsy must still be maintained. We can also see how difficult it is to assert even after prolonged observation in clinical cases that these localized convulsive seizures are not dependent on some organic change in the cortex.

The study of this subject and the review of the literature and the experience of most of us will lead us to the conclusion, however, that there are undoubtedly cases of localized convulsions which are not produced by organic lesions, and this conclusion will have an important bearing on the subject of surgical interference in epilepsy. The question then arises, where can we draw the line, between organic cases which might be benefited by an operation and those cases where an operation would not only be useless, but might damage the patient.

In considering localized convulsions we must in the first place draw a sharp line between convulsions of a local character which precede and those which follow the loss of consciousness; we must also distinguish in the first group between such cases in which there is no loss of consciousness at all, at any time during the history of the case, and those cases in which either in the beginning or later during the course of the disease the local convulsions become general and are attended with the loss of consciousness.

I will take the second group first. Whenever the loss of consciousness occurs first and convulsions follow, limited to a small group of muscles, the cases are undoubtedly epileptic in character and are not Jacksonian in type. These cases are not numerous and should be grouped under the heading of petit mal moteur. It is at times difficult to distinguish such attacks from hysteria. Lately I saw such a case develop on the basis of hysteria.

Miss B., æt. fifty-six, always nervous, about one year ago, after an attack of grippe, began to have stomach symptoms, loss of appetite, intestinal fermentations and gradual emaciation, with occasional vomiting. Six months ago a brother died with hemorrhage from both stomach and bowel. On the day of the funeral patient began to vomit and has continued to do so ever since. Three months ago patient fell and struck the left forehead and temple against the marble floor of the bath. One month later began to have attacks, during vomiting, viz.: loss of consciousness, pallor of face and turning of head over to left shoulder; the loss of consciousness occurs first. At times has had as many as six such attacks per day. It seems to me that we have here a case of traumatic epilepsy with localized convulsions, but not in the sense of Jacksonian epilepsy, but a case of genuine epilepsy, traumatic in origin, engrafted on a neurotic basis, but with localized convulsive seizures.

In other cases of genuine epilepsy we have similarly limited convulsions.

Lilly L., æt. twenty-one, has had epilepsy for six years. The attacks begin without an aura, always with sudden loss of consciousness. The convulsion is usually limited to the left arm. If the attack spreads it involves the left side of face and then the right leg.

This patient has been under observation for two years and the most careful examination has always been negative.

In some cases of genuine epilepsy we have only occasionally a local spasm, say of an arm or the face, the convulsions usually being of a general character.

In other cases of petit mal, which usually have no convulsions, we have attacks occasionally in which there is a local spasm of the arm. Other cases of genuine epilepsy will occasionally have an attack in which, during a few seconds preceding consciousness, an attempt is made to perform a definite act requiring coördination of movement, such as an attempt to brush the hair, but these attacks occur rarely, the usual attack being of the ordinary genuine epileptic type. All of the above variations and irregularities are simply manifestations of genuine epilepsy. It seems to me that the condition of the cortex in the foregoing cases can be compared with those cases of genuine epilepsy in which the epileptic convulsion is followed occasionally by a monoplegia, hemiplegia, aphasia, deafness and other losses of function of the cortex of a focal character. They are so-called cases of abortive epilepsy. The fact that the loss of consciousness precedes the localized convulsion proves them to be genuine epilepsy. Why the convulsion when once begun does not spread is difficult to explain. We may, however, remove these cases entirely from the realm of Jacksonian epilepsy and therefore out of the province of surgery.

In these cases, a most careful examination at various times and a prolonged observation, as well as a careful consideration of the previous history of the case, failed to reveal any evidence of any local change in the cortex which might account for the post-epileptic loss of function. They seemed to present as far as any organic lesions were concerned an absolutely normal condition of the brain.

I mention these cases only to call attention to the fact that even in genuine epilepsy we may have motor manifestations limited to certain areas of the cortex, either always or at times without any apparent reason and with an entire absence of gross organic changes in the cortex, as far as our present methods of physical diagnosis will enable us to determine, during life.

I will now pass on to a group of cases which, either on account of localized convulsive seizures or on account of the onset of the attack with disturbance of function of one of the special senses, I look upon as idiopathic focal epilepsy.

I will begin with the history of two adults who have had but a single attack without any recurrence:

CASE IV. Mr. McK., æt. 24, was examined in March, 1895. He has always been nervous, but has never been seriously ill. On the day before the examination while standing at his door he was seized with a sense of numbness in the left leg which quickly spread to the left arm, at the same time there was a cramp in the arm and leg, he fell, remembers that he was assisted to a couch and then lost consciousness. There was no general convulsion. After regaining consciousness, he found that the left side of the body was paralyzed. The loss of function disappeared in a few hours. Patient came to Cincinnati, a distance of forty miles, by rail, on the following day.

Examination, negative with the exception of neurasthenia. This patient remained under close observation for months, was seen at intervals for years and was again seen a few weeks ago, without a second attack and with no treatment whatever except for neurasthenia.

Résumé.—A local convulsion beginning with numbness, in left leg and arm followed by a spasm, then unconscious with a subsequent passing paresis of the left arm and leg. After thirteen years no recurrence.

CASE V. Mary K., æt. 31, has been under my care for neurasthenia for a few months. Always has been nervous, from overwork, as a clerk.

On November 19, 1906, while at work and in a pleasant frame of mind, suddenly found a difficulty in speaking, could not answer a customer, almost immediately there followed a spasm in the right arm and leg and then she fell, in a state of unconsciousness, was removed to her home.

Examination two hours later: Patient is crying, hysterical, afraid that she is going to die.

Examination reveals still some disturbance of speech of motor character and some weakness of the right arm. The right leg is normal. Sensation and reflexes of right side of body normal. Pulse 96, temperature normal. A few days later patient was back at work.

A second examination three weeks later failed to show any evidence of any organic disturbance of brain.

Patient is well to-day and has never had a recurrence.

Résumé.—A sudden attack beginning with motor aphasia, followed by a localized spasm of the right arm and leg, unconsciousness, then a subsequent partial aphasia and weakness of the right arm, passing off in a few hours. No recurrence in eighteen months. While both these individuals were neurasthenics, I am convinced that the attacks were not hysterical in character, but were due to a temporary and sudden irritation of the cortex of the brain, beginning on a small area and spreading over the entire cortex of one hemisphere. These individuals are not epileptics, but have had a localized spasm of cortical origin, functional in character without a single recurrence.

From the history of the following cases, the manner of the onset of the convulsion and the spread of the seizure with secondary loss of consciousness, we would think at the first examination to have typical cases of Jacksonian epilepsy before us.

Case VI. W. C., æt. 14. Parents healthy, has always been well, never seriously ill. Attacks began in 1895. Patient was seized with a convulsion of the left arm which rapidly spread over entire left side. Then lost consciousness. The convulsions then became general. Has had three attacks of this character.

Examination.—Well developed boy, bright, quick, with good memory.

Examination negative.

In the past thirteen years patient has grown into manhood, over six feet tall, very strong and muscular. He never has any headaches, vomiting or vertigo. Still has occasional attacks, which are possibly brought on by taking a few glasses of beer. The attacks, however, invariably begin in the left arm, extend over left side of body and then loss of consciousness, after which the attacks become general.

Case VII. J. H., æt. 13. Parents neurotic. Never has been sick until six years of age, sudden onset of epileptic seizures. The attacks always begin with tonic and clonic spasms of the right facial, then falls, becomes unconscious and has a general convulsion. Attacks come on during sleep with a scream, but never knows when he has an attack. Often has as many as six in one night and has had twenty per day.

Examination Six Years after Onset of Trouble.—Strong well-developed boy of average intelligence. Physical development good. Examination in every respect negative.

Five years later patient's condition remains unchanged. He has grown into manhood and is in every way normal except for the convulsions.

Résumé.—Healthy young man, epileptic attacks for thirteen years. Onset, always in the region of right facial, no change in the character of attacks since first one. No paralysis.

CASE VIII. C. K., æt. 10, girl, parents normal. In infancy always had " spasms " when sick with a fever. For the past six years has had convulsive seizures, averages one attack per week, has seizures both night and day. At times as many as eight to nine in one night.

The attacks always begin in right hand before they become general. Also has attacks of petit mal.

Examination.—Well-developed girl, stands well in her class.

Examination negative, no weakness or exaggerated reflexes in the right arm, no change in patient at time of writing.

Résumé.—Case of six years standing, local convulsions of right arm usher in each attack of general epilepsy.

CASE IX. The following case has not been under observation long enough (twelve months), but on account of the absence of all symptoms between attacks and the negative character of the examination is of great interest.

John A., æt. 43. Farmer, has three healthy children.

Trouble began ten months ago. Patient complains of a subjective sensation of numbness in the right hand. " The arm has not the right feeling." Attacks begin with scintillations, " sparks," before the eyes then a sensation of tingling in the right side of face, right arm and right leg, loses his ability to talk, " seems to have no mind at all." There is no feeling in the arm and leg, drops whatever he may have in his hand and he can't walk. Never any spasms. He has never during the twelve months lost consciousness. Between attacks there is no headache, no vomiting, no vertigo, there is no loss of strength in the arm or leg.

During the attack has palpitation of heart. Duration of attack two minutes. Frequency. At times several attacks per day, often only one or two attacks per month.

Examination.—Medium height, average development. Intelligence normal. No albuminuria. There was found no change in the optic nerve, no hemianopsia, no loss of strength on right side, no objective disturbance of sensation in any of its qualities and no aphasia, no exaggeration of reflexes.

Résumé.—Attacks, periodic in character affecting entire right side of body, with loss of speech, loss of muscle power and subjective disturbance of sensation, beginning in face and extending to whole right side of body. Typically Jacksonian in character.

CASE X. The next case is somewhat similar in one respect, the numerous attacks limited to attempts at speaking, followed by loss of consciousness and at times general convulsions. Under observation three years.

Miss T., æt. 30. Music teacher, unmarried. As a child had diphtheria and scarlet fever. In the past five months has had five general convulsions with loss of consciousness. One attack began in the right arm and was followed instantly by loss of consciousness. All attacks begin with speech disturbance, numbness and spasms in tongue and lips, during which patient mutters unintelligently and almost immediately loses consciousness. Patient is conscious for a second or two after the onset of the attack. She is conscious of an effort to speak and thinks that she is calling loudly for help, at times the attack passes over in a few seconds, at other times there follows a general convulsion. Patient has frequent attacks of "speech trouble," as described above, which she says she can stop by taking a drink of water, always carrying a small bottle of water with her wherever she goes.

Suffered much with headache. But never has vomiting or vertigo.

Examination.—Negative in every respect, except that right pupil was little larger than left. Patient has been under observation for three years with no change.

CASE XI. The following case presents a feature of local disturbance which is unique.

Otto W., æt. 24, single, draughtsman. Family history negative. Has never been seriously ill.

Onset of trouble five and a half years ago. All attacks begin with a subjective feeling of movement in the right eyeball; at times a loss of vision in the right field (right-sided hemianopsia) precedes the loss of consciousness. This is followed at once by loss of consciousness. Some attacks are limited to the above sensations of a movement in the right eyeball with a "flash of thought through his head" which he cannot recall after the attack.

In the majority of instances the loss of consciousness is followed by a general convulsion, biting of tongue, etc.

Examination.—Mental condition slightly morose and depressed. The examination is in other respects negative.

The following case presents a combination of disturbance of sense of taste, which precedes loss of consciousness, with local convulsions of the right arm and face.

CASE XII. P. H., æt. 17. Has never had any acute illness which could have caused present trouble.

Had first spasm when a year old, one each year until six years old, then none for four years. When a child the right arm was paralyzed after an attack. Up to a few years ago all attacks were as follows: First there is a sensation of having a nasty taste in the mouth. "The taste of worms" nauseating in effect; the boy then runs to washstand or waste-jar and begins to expectorate saliva from the mouth, almost immediately there is a spasm in the right arm and face. Then follows loss of consciousness, is not sure that the turning of head to right and convulsion of face and arm precedes the loss of consciousness. But the convulsion always begins with the spasm in the right side of face, turning of head to right, then right arm and leg are affected and then the convulsion becomes general.

For the past five years attacks begin with the exclamation, "Oh, stuff is coming up," which is quickly followed by a loss of consciousness and a general convulsion. Patient states that the above cry is always the result of a foul taste in the mouth which seems to rise from the throat. Sometimes patient has attacks only of disturbance of taste with loss of consciousness.

The majority of his attacks now are those in which he has the conscious feeling of a vile taste in the mouth, then expectorates. There is a flow of saliva and then loss of consciousness and a general convulsion. At times, however, the convulsion begins in the right side of face, head turns to right, then a convulsion in the right arm and leg, after which the convulsion becomes general.

Between attacks he is well, has no headache, vomiting or vertigo. Sometimes has no attacks for weeks. Has had sixteen spells in the last thirty-six hours.

Examination.—Mental condition is sluggish, otherwise negative, including sense of taste and smell.

In the foregoing group of cases I was very careful to include only such which were entirely negative as far as any objective examination can reveal the presence of slight organic changes in the cortex. I do not overlook the fact that these changes may be present in the neighboring latent or silent areas of the frontal, parietal or temporal lobes. Horsley operated on five cases of the above character and in two found organic cortical lesions. Years ago I had a case of so-called idiopathic epilepsy operated upon and found that a small diploic vein had worn through the inner table and had caused a perforation of the dura and an irritation of the cortex. However I believe that, unless we wish to become Nihilists in our clinical work, we must hold as a working proposition, hard and fast to the rule, that if the objective examination is negative, especially in cases which have existed for years, there is no gross lesion of the cortex. Although therefore, as to the subject of Jacksonian epilepsy, we are often standing on uncertain ground, we have some good rules for guidance to distinguish idiopathic from Jacksonian seizures.

There are two points of distinction between the so-called idiopathic local epileptic convulsions and the symptomatic Jacksonian.

The idiopathic partial epilepsy shows no progression. In all the cases which I have cited above, there has practically been no change in all the years during which they have been under observation. They always begin in the same way and the secondary paresis has never become permanent or progressed to a real paralysis. In the symptomatic Jacksonian attacks, however, we have a steady progression. The symptomatic spasms have a gradual beginning; at first almost unnoticeable, muscular twitchings occurring at long intervals. Then a gradual increase in severity, number and groups of muscles involved until after weeks, months or years they attain their height and maximum. Then again the secondary paresis in symptomatic Jacksonian epilepsy sooner or later becomes permanent, progressive, persisting during the entire interval between the attack. In the second place, in the idiopathic Jacksonian seizures, the objective physical examination is negative, whereas in the symptomatic form, sooner or later we will find objective signs, which point to an organic change in the cortex.

The symptomatic form is less apt to be nocturnal than the idiopathic, but since symptomatic Jacksonian attacks do arouse patients from sleep, we cannot rely very much on this point, although the presumption would be in favor of the idiopathic form.

Are we justified in looking upon cases of the above character as idiopathic epilepsy? Does the fact that we are unable to find on physical examination any deviation from the normal prove that there is no gross change in the cortex of the brain?

The history of epilepsy abounds with cases of supposed idiopathic epilepsy in reality caused by gross pathological lesions, even brain tumor, which were not suspected during life. Horsley reports that in two out of five cases of idiopathic local epilepsy, he found on operation gross lesions of the cortex. Until we have careful clinical investigation on the line followed by Redlich, we cannot but hold that for practical working purposes, when the physical examination is negative, there is no gross lesion of the cortex. Investigating the widely promulgated theory, that a vast number of cases of youthful epilepsy are caused by cerebral palsy in early childhood, Redlich carefully examined 150 cases of genuine epilepsy in young subjects. Of these, three showed manifest evidence of hemiplegia and he excluded them as not being cases of genuine epilepsy, but cases of cerebral palsy with epilepsy. He insists that we must not look for marked physical signs caused by gross lesions, but slight deviations produced by finer anatomical changes. He found slight evidences of paresis on the part of the cranial nerves and of the extremities, brought out by the use of the dynamometer, rapidity of exhaustion of one or both limbs on one side as compared with the other, and differences in the reflexes, especially the skin reflexes, of the two sides. He also found not only passing, but more or less permanent states of weakness or paresis of extremities after an attack.

Basing his opinion on these clinical findings, he came to the conclusion that 40 per cent. of cases of genuine idiopathic epilepsy show the presence of organic change in the cortex, in another 15 per cent. the indications were doubtful, and in 45 per cent. the cases were negative.

Research of this character is of value, if only in the sense that when painstaking investigation shows an absence of all clinical signs, we can presume that gross cortical changes are not present.

Two opinions can readily be held as to the cellular changes found in the cortex. Clark and Prout's investigations as to the changes in structure of the ganglionic cells of the cortex have been widely quoted as having an important bearing on the pathology of epilepsy. Collins in his case of excision of the cortex found microscopic cellular changes in an apparently normal cortex, and the recent publication of Jelliffe's experiments indicate that cellular changes of a widespread character may underlie these apparently idiopathic cases. Osler, on the other hand, believes that these minute changes can result from and be caused by the epileptic seizures themselves, resulting from the circulatory changes which accompany the attack. This is further supplemented by the angioneurotic theory of Quinke, who supposes that a local spasm of a small vessel may cause the focal symptoms in local idiopathic epilepsy. Perhaps the explanation may be found along the lines of work of Spiller, McCarthy, and others, who found minute areas of softening, perhaps the result of capillary embolism in uremic and arteriosclerotic conditions. Redlich, Chaslin, Bleuer, Buchholz, Altzheimer, Weber, Orloff, believe that we may have localized increase of the neuroglia tissue. On the other hand, Weber has shown that this localized increase of the neuroglia tissue does not bear any constant relation in its cortical location to the point of origin of the muscular spasms.

That the lesion, whatever it may be, is not necessarily progressive is shown by the course of the majority of the above-cited cases.

In considering the surgical aspect of these cases, I wish to confine myself entirely to the discussion of cases of the above type, namely: localized idiopathic epilepsy.

It resolves itself therefore into two questions, first, the question of an exploratory operation, which necessarily means the opening of the dura mater and the electrical examination of the cortex, and secondly the excision from the cortex of the area corresponding to the seat of the initial convulsion.

The surgical opinion on this subject is pretty well expressed by Roswell Park and J. Chalmers Da Costa.

Roswell Park, in speaking of the surgery of epilepsy, divides the appropriate cases into three groups: (1) Those presenting reflexes from peripheral irritation, (2) those presenting gross anatomic lesions within the cranium, (3) cases of so-called genuine epilepsy of non-traumatic origin presenting localizing phenomena.

In our discussion, the third group alone is of interest. It covers a wide group of cases and makes no distinction as to clinical findings.

J. Chalmers Da Costa says: " That a correct decision cannot be reached intuitively, a conclusion must not be jumped at. An opinion to be of value must emanate from some one who possesses a scientific knowledge of epilepsy and has examined the patient with painstaking care." Concerning idiopathic focal epilepsy he says: " If from the very start the convulsive attacks have a local beginning and if the surgeon has seen the case within two years of the first attack, it is proper to trephine and excise the irritated portion of the cortex. This is true whether the convulsions remain partial or spread to the rest of the body, whether consciousness is lost or not. He advocates operations in all cases whether the convulsions are local in the beginning as well as in cases of general convulsions which afterward assume a local character, if the attacks are very numerous, are uninfluenced by ordinary treatment and in any way threaten life. On the other hand, Horsley has lately expressed the opinion that idiopathic focal epilepsy is not amenable to surgical treatment.

In considering the advisability of surgical interference, therefore, we must be absolutely satisfied that we have ruled out reflex epilepsy, the motor aura and hysteria. According to Starr, 23 per cent. of all cases show a local or focal origin.

The first point to be considered is the exploratory operation. This involves necessarily the opening of the dura, the examination of the cortex, both mechanically and electrically. In the first place the operation itself has quite a large mortality in idiopathic cases where there is no intracranial pressure. In cases of this kind Horsley lost one case out of five, viz.: 20 per cent. Matthiolius' statistics show a mortality of 24 per cent. When we consider that the disease for which the operation is performed is mild and rarely terminates fatally, the above results should make us halt and consider. As to the actual mortality in operations in a large number of idiopathic cases of focal epilepsy, I have not been able to find statistics. If the cortex is not excised it cannot be examined without causing some local damage, and this damage is not necessarily confined to the immediate area involved, but the surrounding cortex must necessarily be explored; then we always leave a local imperfectly repaired dura mater, danger of adhesions between the cortex and membranes, and finally a large bony defect. In a word, the operation itself substitutes for at most microscopic defects of the brain gross surgical defects and local contusions and hemorrhages in the cortex. Rasumowsky, however, states that not one of his seven cases was damaged mentally or physically by the ablation of the cortex.

Let me introduce a case here which serves to illustrate the dangers of surgical interference.

Case XIII.—Mrs. W., æt. 34, married, six children.

Has been of a nervous disposition; some years ago had an injury, a piece of iron fell from a tree and struck her on the left side of head. Was unconscious for a few minutes. Later complained much of headache and developed some weakness in the right side of body, with loss of sensation.

On the supposition that there was a depression or splintering of the inner table, patient was trephined over the left parietal region. The operation was successful, but no defect of bone, except an unusual thickness, was found.

The symptoms remained unchanged and a second operation was performed, another section of bone was removed. The headache and weakness and loss of sensation on the right side remained unchanged. Patient became pregnant and during delivery had a severe general convulsion. Gradually she lost vision in left eye.

Later on she developed convulsions in the right arm, which extended to right side of face, and then became general after losing consciousness.

A third operation was performed, a large area of bone removed but nothing was found except a normal cortex.

After third operation patient had more convulsions, the vision of left eye was entirely lost and the headaches continued; there was a weakness of right arm and leg with loss of consciousness. Patient was brought to Cincinatti in this condition and placed in the Good Samaritan Annex for observation.

There is no vomiting nor vertigo, constant headache for past seven weeks.

Examination.—Mental condition very changeable, exaltation varying with depression.

No optic neuritis, left papilla unchanged. But patient cannot see with left eye. Left pupil is larger than right.

Loss of sense of smell on right side, weakness and increased reflexes of right side, but no Babinski, Oppenheim, or ankle clonus. Sensation in all of its qualities is lost on right side of body, including the face. There is hemianesthesia, hemianalgesia, thermanesthesia, loss of muscle sense and astereognosis.

After ten weeks all the symptoms disappeared with the exception of fainting spells; since her return home eighteen months ago, patient has born another child and has had three general convulsions.

This case is instructive in several respects. The paralysis of the right side of the body was hysterical in character and was presumably the same before the operation; at least, one of our best neurologists who saw her before and after the third operation was convinced after the operation that the paralysis and loss of sensation were hysterical. It yielded to suggestive treatment. In the second place, the epileptic seizures occurred only after the second operation; there is no doubt as to the genuine character of the epileptic seizures, and, to say the least, if the original injury was the cause of the epilepsy, the extensive surgical interference failed to find a cause and failed to prevent the development of both the Jacksonian and genuine epileptic attacks. My judgment of the case is, that the surgical interference was futile and ineffectual, to say the least.

What is the experience as to excision of the cortex? We must remove from consideration at once all these cases whose point of origin seems to be in the special senses, especially those with initial visual and speech symptoms. This leaves for consideration only those with initial sensory or motor symptoms. The sensory cases can also be dismissed on account of our imperfect knowledge of sensory localization of the cortex and the impossibility of verifying the center involved unless we follow Cushing's suggestion and use no anesthetic. However, there could not be sufficient accuracy to justify cortical excision in these cases. This narrows down the field of operation to cases with motor manifestations. We know that some of the earliest experiments in epilepsy were those of Hitzig, who produced epilepsy in dogs by excision of portions of the cortex of the motor area. Jacksonian epilepsy followed, but soon the convulsions became general, losing the Jacksonian type, beginning with initial loss of consciousness. Moreover, the experiments of Munk and Sherrington on apes has been apparently verified in man. While most operators have reported a quick disappearance of the paresis and paralysis following excision of the motor area, Da Costa says: "That the finer movements of the fingers are never regained."

Another question arises in these cases; even though we should decide to extirpate the cortex, we can never be sure that the source of irritation is the region indicated by the origin of the convulsions.

Drivet (These de Bordeaux) collected thirty-five cases, from the literature, of Jacksonian epilepsy beginning in the face or arm; out of thirty-five cases twenty-six times the lesion was in the motor area, but not always in the area indicated (viz., the face area) nine times outside of the motor area and twice in the cerebellum.

Cramer and Weber publish a typical case of Jacksonian epilepsy caused by atrophy of the cerebellum.

The above are the objections to the excision of the cortex based upon experimental and clinical observations. It seems to me that any one of the above cases, with a normal condition of the hands and face, for instance, is better off than if these regions were left permanently paretic or deprived of the finer functions of coördinate movement.

Now what are the surgical results in the above class of cases as gleaned from tables of statistics?

There are very few cases on record of excision of the cortex in idiopathic localized epilepsy. Bergman, Dejerine, Braun, Horsley and Gowers, Raymond, Donath, Putnam, Pick and others are opposed to the operation, Collins and Sachs are in favor of it. Da Costa says that only one per cent. of cases are cured. Braun collected thirty cases of ablation of the cortex in traumatic cases, thirteen were reported cured, but only three had been observed more than three years. Braun says that there is not a single case of operation for focal idiopathic epilepsy with ablation of the cortex, which has been under observation more than three years (up to 1899).

Mattheolius has been able (up to 1899) to collect fourteen cases of the above character, viz., idiopathic focal epilepsy, in which the cortex was excised with but one cure, and this case had not been observed more than two years.

Rasumowsky publishes two cases with brilliant results. These, with Collins' case, certainly hold out some hope. Nast-Kolb's tables bring up the statistics to 1904; of eighty-six cases, fourteen were apparently cases of idiopathic focal epilepsy in which the cortex was removed, in eleven of the cases after electrical investigation. In five of these cases the freedom from convulsions lasted for from three to twelve years, a result which may once more raise our hopes.

Raymond, basing his views upon a review of thirty-one collected cases of Jacksonian epilepsy with only three cures, is opposed to the operation. He bases his opposition on the presence of the epileptogenic condition being present beyond the seat of origin in the cortex, a widespread change, which is present in a quantitative manner to a greater degree in the affected zone, but nevertheless present over the entire cortex. Horsley says that no general laws can be laid down, that it is impossible to argue as to the surgery of epilepsy except as to individual cases.

My own judgment, based upon the experience of cases operated upon and those not operated upon, both being observed for a long time, is, that the presence of localized convulsions alone should never be an indication for an operation. These individuals, in idiopathic focal epilepsy, already possess the epileptogenic changes (Jolly) of the cortex in areas beyond the first focus of discharge. The removal of that focus will not change the remaining areas of the cortex. When the attacks have been general and frequent, with complete loss of consciousness, no cure can be expected from any surgical operation. If, after prolonged observation, there is no marked progress of the disease and the case is more or less amenable to medical treatment and no localizing physical signs develop, I believe that no surgical measures should be resorted to.

If, however, the case rapidly becomes worse in the sense of numerous attacks of a violent character and the individual's life work is handicapped by the disease and if the medicinal treatment fails to give relief, an exploratory operation should be recommended in the hope of finding some local disease of the brain or its coverings. Whether the cortex should be removed is a debatable question, although the statistics of Nast-Kolb would seem to be favorable to the operation. In gross lesions, I believe we can say that the number and severity of the attacks are diminished and made more amenable to treatment after the operation.

Under the above restrictions Da Costa advocates the ablation of the cortex in idiopathic focal epilepsy, stating that after the operation the attacks are less numerous and more amenable to treatment, even though no absolute cure is effected.

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